AMENDMENTS TO THE CLAIMS

1-26. **(Canceled)**

27. **(Currently amended)** A method for reducing memory dysfunction associated with damaged hippocampal tissue in a mammal exhibiting memory dysfunction and caused by permanent or transient global ischemia, comprising the steps of: determining the existence of memory dysfunction, and administering to the mammal a morphogen comprising a conserved C-terminal seven-cysteine skeleton that is one or more of the following:

(a) at least about 60% identical to residues 330-431 of human OP-1 (SEQ ID NO: 2); and

(b) at least about 70% homologous to residues 330-431 of human OP-1 (SEQ ID NO: 2),

thereby reducing memory dysfunction associated with damaged hippocampal tissue in the mammal.

28. **(Previously Presented)** The method of claim 27, wherein said morphogen stimulates synapse formation between hippocampal neurons.

29. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 30-292 of SEQ ID NO:2.

30. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 330-431 of SEQ ID NO:2.

31. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 48-292 of SEQ ID NO:2.

32. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises the amino acid sequence of SEQ ID NO:2.

33. **(Canceled)**

34. **(Currently Amended)** The method of claim 28, wherein said morphogen comprises a mature form of human OP-1, defined by residues 293-431 of SEQ ID NO: 2.

35. **(Currently Amended)** The method of claim 29, wherein said morphogen comprises a mature form of human OP-1, defined by residues 293-431 of SEQ ID NO: 2.

36. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-2 polypeptide.

37. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-5 polypeptide.

38. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-6 polypeptide.

39-42. **(Canceled)**

43. **(Previously presented)** The method of claim 27, wherein the morphogen is administered by intraventricular administration.

44. **(Previously presented)** The method of claim 27, wherein the morphogen is disposed in a biocompatible microsphere.

45. **(Canceled)**

46. **(Currently amended)** A method for reducing memory dysfunction associated with damaged hippocampal tissue in a mammal exhibiting memory dysfunction, comprising the steps of: determining the existence of memory dysfunction, and administering to the mammal a morphogen comprising a conserved C-terminal seven-cysteine skeleton that is one or more of the following:

(a) at least about 60% identical to residues 330-431 of human OP-1 (SEQ ID NO: 2); and

(b) at least about 70% homologous to residues 330-431 of human OP-1 (SEQ ID NO: 2), wherein the damaged hippocampal tissue is damaged by ibotenic acid, ammonia and formaldehyde.

47. **(Canceled)**

48. **(Currently amended)** A method for reducing memory dysfunction associated with damaged hippocampal tissue in a mammal exhibiting memory dysfunction, comprising the steps of: determining the existence of memory dysfunction, and administering to the mammal a morphogen comprising a conserved C-terminal seven-cysteine skeleton that is one or more of the following:

(a) at least about 60% identical to residues 330-431 of human OP-1 (SEQ ID NO: 2); and

(b) at least about 70% homologous to residues 330-431 of human OP-1 (SEQ ID NO: 2),

wherein the ~~mammal is afflicted with~~ damaged hippocampal tissue is damaged by malnutrition, glucose metabolism disorder, or anorexia.

49-50. **(Canceled)**

51. **(Previously presented)** The method of claim 48, wherein the mammal is afflicted with malnutrition.

52. **(Previously presented)** The method of claim 48, wherein the mammal is afflicted with a glucose metabolism disorder.

53. **(Previously presented)** The method of claim 48, wherein the mammal is afflicted with anorexia.